measured by IVUS in vivo in humans. Adventitia is classically non-analysable during routine IVUS examination but is on the other hand properly detectable under the conditions of observation here described and, at least in some segments, can be looked out for. Given the particular anatomical pattern being studied here measurement was from inner border (external elastic membrane) to outer border; this is a limitation that reduces the accuracy of our measurements, as outer border data are well known to depend on the radiofrequency envelope thickness and to be affected by the gain. This explains why the minimum distinguishable distance between two finite points should at best correspond to the system's axial resolution (190 μm at 30 MHz). These acoustic limitations account for the 158 μm offset from the regression slope in our findings. Adventitia has recently been reported visualised by high frequency epicardial echocardiography during heart surgery.[4] The periarterial halo that the authors identify as being the adventitia was significantly thicker in case of atherosclerosis detected on angiography than in an angiographically normal artery (0.92 (0.2) mm *v* 0.54 (0.2) mm; $p < 0.001$), but unfortunately this was not correlated with histological studies. These values are twice as high as our own, probably because of the poorer resolution of their measurement system: 6–15 MHz. Some pathological studies, however, do confirm our finding of significant adventitial thickening in case of atherosclerosis.[2–5] We observed no significant difference in adventitial thickness according to the type of arterial remodelling. The adventitia is widely recognised as being implicated in the constrictive remodelling subsequent to balloon angioplasty,[3] but the findings are much less well established in the case of native atherosclerosis. In a recent autopsy series, Burke *et al*[5] found that negative remodelling appeared to be associated with thicker adventitia. On multivariate analysis, however, the involvement of the adventitia in the type of remodelling proved insignificant. Very recently, Moreno *et al*[2] reported adventitial inflammation in aorta sections associated with the largest intraplaque lipid zones, especially in association with the largest atheromatous plaques.

The coronary adventitia is often detected in the LAD artery and is shown to be easily and reliably analysable on IVUS with an increased thickness in the case of coronary atherosclerosis.

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IMAGES IN CARDIOLOGY

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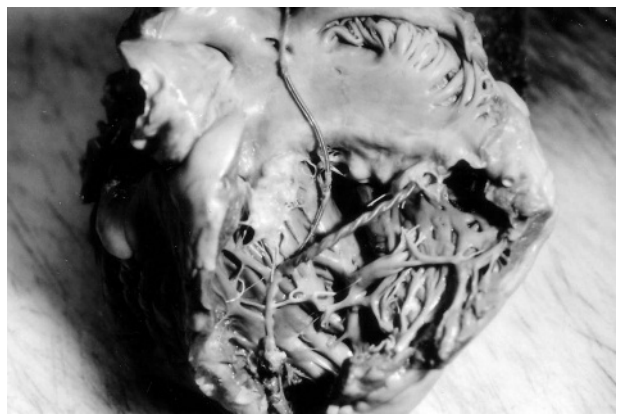
An unusual post mortem illustration of twiddler's syndrome

This photograph shows a postmortem heart. The right atrium and right ventricle have been opened, showing the tricuspid valve. A permanent pacemaker lead descends from the superior vena cava to the right atrium and thence the right ventricle. A loop of twisted pacemaker lead of at least 10 coils is shown orientated left and superiorly. There was loss of electrical insulation proximal to the twisted segment. At fresh postmortem examination, manual torsion of the wire at the left infraclavicular pulse generator site resulted in transmission of the twist right down to the right ventricle despite the presence of numerous endocardial adhesions around the wire.

The patient, a woman aged 85, had received a unipolar VV1 pacemaker six years previously for third degree atrioventricular heart block. Six months later re-programming for pocket twitch was required. Eighteen months after that, further pocket twitch prompted reoperation, finding the pulse generator turned over, exposing pectoral muscle to its non-insulated aspect. The pulse generator was replaced with another firmly stitched down. Four years later she presented with heart failure, atrial flutter and intermittent failure to capture the ventricle. A major cerebral infarction caused death.

Usually, twiddler's syndrome results in loops of wire at the pulse generator site. This unusual case shows how, despite multiple adhesions, torsion can be transmitted to within the heart. As usual, the patient stoutly denied twiddling.



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